PATENT
Attorney Docket No.: 2307AA-031220US
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IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re application of:

Steinunn Baekkeskov *et al.*

Application No.: 08/838,486

Filed: April 7, 1997

For: IMPROVED METHODS FOR THE DIAGNOSIS AND TREATMENT OF DIABETES

Examiner: G. Ewoldt

Art Unit: 1644

REQUEST FOR INTERFERENCE UNDER 37 C.F.R. § 1.607 and 1.608(a)

Assistant Commissioner for Patents
Washington, D.C. 20231

Sir:

Applicants request that an interference be declared between the present application and Atkinson *et al.* US 5,762,937 and US 6,001,360 under 37 CFR 1.607 and 1.608(a). It is recognized that the Examiner has said that he is not going to comment on an interference until the claims are indicated to be allowable. Nevertheless, if the enablement rejection is reversed on the appeal, then presumably the Board will consider the art rejection including the question of whether the '937 and '360 patents contain interfering subject matter with the present claims. Therefore, applicants are providing a formal request for an interference at this time.

I. Declaration of an interference is proper under 1.608(a)

(1) Effective Filing Dates are within Three Months

The present application is a divisional of 08/452,053 filed May 25, 1995, which is a continuation of 08/174,550, filed December 28, 1993, which is a continuation of 07/756,207 filed September 6, 1991, which is a continuation-in-part of 07/579,007, filed **September 7, 1990**.

The '360 patent derives from 07/474,598 filed June 7, 1995, which is a continuation-in-part of application USSN 08/219,816 filed Mar. 28, 1994 now U.S. Pat. No. 5,762,937, which is a continuation of USSN 08/093,821, filed Jul. 19, 1993, now abandoned, which is a continuation of USSN 08/007,406, filed Jan. 22, 1993, now abandoned, which is a continuation of USSN 07,569,324, filed Aug. 17, 1990, now abandoned, which is a continuation-in-part of USSN 07/427,051, filed Oct. 15, 1989, now abandoned, which is a continuation-in-part of USSN 07/283,633, filed Dec. 13, 1988, now abandoned. However, the earliest two filed of these applications, '051 and '633, contain no disclosure relevant to the present claims, specifying methods of using GAD as an analytical reagent. It is noted that the '051 and '633 misidentified the autoantigenic component of the pancreatic 64 kDa antigen as being a serine kinase, rather than GAD. Thus, the '360 and '937 patents at best derives priority from the '324 application filed **August 17, 1990.**

Accordingly, the effective filings date of the present application and the earliest conceivable effective date of '360 and '937 patents are only three weeks apart, within the three month period specified by 1.608(a).

(2) Basis for Judgment

Attached is a declaration stating that Applicants have a basis for judgment relative to the '360 and '937 patents.

II. Requirements of § 1.607 are satisfied

(1) Identification of the patent

US 5,762,937 and US 6,001,360

(2) Proposed Count

Applicants propose a three element phantom count as follows:

Claim 31 or claim 62 of the present application or claim 1 of US 6,001,360.

Claim 31 and claim 62 are included in the alternative because there are additional grounds of rejection pending against claim 62 that are not applicable to claim 31. Thus, it is possible that the enablement rejection will be reversed for claim 31 but not claim 62.

3. Correspondence of Patent Claims to Count

Applicants submit that each of claims 1-2 of the '360 patent corresponds or substantially corresponds to the count. Claim 1 of '360 corresponds exactly to the third element of the count. Claim 2 of '360, directed to recombinant GAD, corresponds substantially in that techniques for recombinant expression were well known in 1990. Claim 1 of US 5,762,937 differs from claim 1 of the '360 patent in two respects. First, claim 1 of '937 is directed to GAD whereas claim 1 of '360 is directed to GAD or fragments thereof. The recital of only one of two alternative embodiments does not confer patentable distinction. Second, claim 1 of '937 recites that administration of GAD reduces the severity of insulin dependent diabetes whereas claim 1 of '360 recites that administration prevents or delays the development of clinical symptoms of GAD. However, given the mechanism of GAD, these are simply semantic differences. GAD is an autoantigen whose administration induces tolerance to the β-pancreatic 64 kDa autoantigen and hence delays or prevents further destruction of the autoantigen. It follows that GAD acts only on the underlying basis of disease (*i.e.*, destruction of β-pancreatic cells) and does not have any effect on the clinical symptoms except as a consequence of its effect on the underlying disease. Thus, if one prevents or delays destruction of β-pancreatic cells, one also delays or prevents the clinical symptoms that follow from such destruction, or in other words reduces their severity. Claims 2 of '937 substantially corresponds to the count for the same reason as claim 2 of '360.

4. Claims in Present Application Corresponding to Count

Claim 31 corresponds exactly to the first element of the count. Claim 35, directed to a pharmaceutical composition, corresponds substantially to the count in that it is routine to administer a therapeutic agent (in this case GAD) as a pharmaceutical composition. Claims 50 and 51 correspond substantially to the count in that recombinant expression and use of a peptide synthesizer were common practice as of September 1990. Claim 52 corresponds substantially to the count in that the CNS was known to be a source of GAD in September 1990 (see). Claim 56 corresponds exactly to the count. Claim 53 corresponds to the count in that the mechanism of action of GAD of inducing tolerance to the principal autoantigen in insulin dependent diabetes suggests that GAD be administered before complete destruction of the autoantigen (*i.e.*, to prediabetic patients). Most such patients inherently have autoantibodies to GAD. Claims 54-57 substantially correspond to the count for analogous reasons to claims 50-52. Claim 62 corresponds exactly to the second element of the count. Claim 63 corresponds substantially for analogous reasons to claim 50.

5. Support for Claims Corresponding to Count

Attached is a table showing support in the present application and the priority document for all claims from the present application.

6. Requirements of 35 USC 135(b) are met

Claim 31, which corresponds to the count, was amended to its present form in an amendment filed October 28, 1998. This is within a year of the issuance of Atkinson, US 5,762,937 on June 9, 1998. Further, claims 60 and 61, which were copied from US 6,001,360 were filed in an amendment of April 14, 2000, within a year of issuance of the '360 patent on December 14, 1999. Thus, the requirements of 35 USC 135(b) are met.

Content of pending claim in US 08/838,486	Sentence(s) spanning page: lines in 08/838,486	Claim filed with application in 08/838,486	Sentence(s) spanning page: lines in 07/579,007	Claim filed with application in 07/579,007
31. A method for inhibiting the development of insulin dependent diabetes mellitus, said method comprising administering to a patient a therapeutically effective dosage of glutamic acid decarboxylase (GAD).	page 21:lines 22-23	Claim 31	page 18:line 33-page 19:line 1	Claim 31
35. (Twice amended) A composition comprising glutamic acid decarboxylase, which is at least 99% w/w/ pure, in a pharmaceutically acceptable carrier for	p.14, line 14	Claim 35	p. 12, line 33	Claim 35
parenteral administration to a human patient.	page 21:lines 1-7		page 17:line 35-page 18:line 1	
50. The method of claim 31, wherein the GAD is recombinant GAD.	page 12:lines 25-29		page 11:lines 8-12	
51. The method of claim 31, wherein the GAD is synthesized on a peptide synthesizer.	page 12:lines 12-24		page 10:line 32-page 11:line 7	
52. The method of claim 31, wherein the GAD is purified from the central nervous system tissue.	page 9:lines 31-36		page 9:lines 4-7	
53. The method of claim 31, wherein the patient is a prediabetic patient having	page 6:lines 13-22		page 5:line 34-page 6:line 3	

autoantibodies to GAD.				
55. The composition of claim 35, wherein the GAD is recombinant GAD.	page 12:lines 25-29		Page 11:lines 8-12	
56. The composition of claim 35, wherein the GAD is synthesized on a peptide synthesizer.	page 12:lines 12-24		page 10:line 32-page 11:7	
57. The composition of claim 35, wherein the GAD is purified from the central nervous system tissue.	page 9:lines 31-36		page 9:lines 4-7	
59. The composition of claim 54, wherein the GAD65 is human GAD65.	page 11:lines 3-6		page 9:lines 29-32	
62. A method of preventing or inhibiting the development of insulin dependent diabetes, wherein said method comprises administering to a patient at least 99% w/w pure GAD protein or a fragment thereof, which, when administered to the patient,.	page 14, line 14	31	page 12, line 33	31
GAD is 99% w/w pure	page 14:lines 12-14		page 12:lines 32-34	
prevents or inhibits the development of insulin dependent diabetes	page 21:lines 18-22 page 1:lines 29-31		page 18: lines 33-37 page 1 lines 25-27	
63. The method of claim 62, wherein the GAD protein or fragment thereof is a recombinant protein.	page 12:lines 25-29		page 11:lines 8-12	

64. The method of claim 31, wherein the GAD is administered intravenously.	p. 20, line 23		p. 18, line 1	
65. The method of claim 62, wherein the GAD or fragment is administered intravenously.	p. 20, line 23		p. 18, line 1	
66. The method of claim 31, wherein the GAD is administered subcutaneously.	p. 20, line 23		p. 18, line 1	
67. The method of claim 62, wherein the GAD is administered subcutaneously.	p. 20, line 23		p. 18, line 1	

Respectfully submitted,

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